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RECORDINGS OF VISUAL EVOKED POTENTIALS AFTER PHOTOSTRESS IN ARTIFICIALLY INCREASED INTRAOCULAR PRESSURE

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Summary—1. The macular recovery after photostress has been investigated in 30 normal subjects under conditions of experimental ocular hypertension. In 18 subjects monocular visual evoked potentials (VEPs) were recorded after photostress in the left eye (control VEP) and then in the right eye, which was previously subjected to an intraocular pressure (IOP) increase.

2. In the control eyes, the VEP recorded 20 s after photostress presented an increase in latency and a decrease in amplitude. The functional recovery was complete after 80 s. In the eyes with induced ocular hypertension the parameters of VEP after photostress underwent larger changes than in the control eyes.

3. Our results indicate that the VEP recovery time after photostress depends both on retinal photopigment ability of resynthesis and on trophism of the macula papillo-macular bundle system.

4. This test could be useful in the clinical evaluation of the functional state of the papillo-macular bundle, in pathologies like glaucoma.

Key words—VEP; photostress; macula; intraocular pressure; glaucoma.

INTRODUCTION

The mechanism of vision depends upon the state of adaptation of the photoreceptors: a bleaching of a portion of the retina alters the adaptation process with the consequent formation of a scotoma; the return to the normal condition depends on the integrity of the complex pigmented epithelium-photoreceptors, functionally crucial for the resynthesis of macular pigment.

Baillart (1954), in the clinical evaluation of the central retina, suggested to evaluate the macular functionality by measuring the recovery period of visual acuity after dazzling of the macular region with an ophthalmoscope. This test (MPST, macular photostress test) was indicated as an index of the "functional macular reserve".

The MPST was carried out on normal subjects (Severin *et al.*, 1967; Zingirian *et al.*, 1968; Franzone *et al.*, 1985) on diabetic ones (Zingirian *et al.*, 1985; Mosci *et al.*, 1986) and on subjects suffering from glaucoma (Polizzi *et al.*, 1984).

Sherman and Henkind (1988) studied the macular recovery period in subjects suffering from glaucoma and they found that the macular recovery after photostress was altered. With their subjective method (MPST) they could not determine the relative contribution of the different functional layers of the retina to the delay in macular recovery, caused by glaucoma.

An objective method to evaluate the visual function is to record the cortical potentials evoked by patterned stimuli (visual evoked potentials, VEPs) and/or the electroretinographic signals (flash- or pattern-ERG).

Is known that the FERG is originated predominantly in the outer layers of the retina (Armington, 1974) while the PERG is originated in the innermost retinal layers (Maffei and Fiorentini, 1981, 1982; Hollander *et al.*, 1984; Maffei *et al.*, 1985).

For instance it is known that the pattern VEPs and PERG are altered in patients suffering from glaucoma (Sokol *et al.*, 1981; Towle *et al.*, 1983; Wanger and Persson, 1983, 1985; Atkin *et al.*, 1983; Bobak *et al.*, 1983; Howe and Mitchell, 1986; Marx *et al.*, 1986a,b, 1988; Porciatti *et al.*, 1987; Bucci *et al.*, 1988; Mierdel *et al.*, 1988) and maculopathy (Bass *et al.*, 1985; Celesia and Kaufman, 1985).

Lovasik (1983) and Franchi *et al.* (1987), studied the recovery of macular function using the VEP method after dazzling.

Initially, the VEP was measured in the control condition, then macular dazzling was produced and the recovery time was evaluated. Recovery

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time is the time necessary for the VEP to return to the control level. In all normal subjects a total recovery of the VEP morphology was achieved after 60 s. In subjects suffering from maculopathy, the recovery time was noticeably longer. The length of the delay was correlated with the extent of the anatomofunctional changes in the photoreceptor-pigmented epithelium system.

In the light of these results we wanted to evaluate with an objective test:

(a) the recovery time in normal subjects analyzing the variation in VEP and its return to control condition after macular dazzling;

(b) the macular recovery time in subjects with induced ocular hypertension, analyzing the variations of VEP and its recovery after macular dazzling in artificially increased intraocular pressure (IOP); and

(c) the possible differences between the recovery of the macular function after dazzling in normal subjects and in those with induced hypertension, with the aim of finding correlations between the increase in IOP and the recovery of the macular functionality.

SUBJECTS AND METHODS

Recordings of VEP were carried out on 28 subjects.

Only data from 15 subjects (30 eyes) were considered, because in the other cases the electrophysiological recordings presented too many artifacts per trial and therefore the total duration of the single trials exceeded the pre-established VEP and FERG recording time.

It was not possible for us to carry out pattern-electroretinogram (PERG) or focal-electroretinogram recordings because the method we used to increase the ocular tone (application of ophthalmodynamometer) interfered with the recordings of the signals.

Fifteen subjects ranging from 42 to 56 yr of age (average 48.7 $\pm$ 4.9 yr) were examined: the ophthalmoscopic examination did not reveal any pathology of the optic disk or of the dioptric parts. In all subjects, VEPs were recorded after photostress in the left eye (15 eyes), and after photostress plus experimental hypertension in the right eye (15 eyes).

The subjects under examination were seated in a semi-dark room acoustically isolated. The display was surrounded by a uniform field of luminance 5 cd/m^2. The subjects were informed of the type of examination and its diagnostic uses.

In the first group *control VEPs* were recorded using the following method.

The visual stimuli were checkerboard patterns (contrast 70%, mean luminance 110 cd/m^2) generated on a TV monitor and reversed in contrast at the rate of 2 reversals/s. At the viewing distance of 114 cm the check side subtended 15 min of visual arc and the screen of the monitor subtended 25 deg. The stimulation was monocular, after occlusion of the other eye.

Cup shaped electrodes of silver–silver-chloride were fixed with collodion in the following positions:

—active electrode in O_z, O_1, O_2;
—reference electrode in F_{pz};
—ground in left arm.

The interelectrode resistance was kept below 3 kΩ. The bioelectric signal was amplified (gain 20,000), filtered (band-pass 1–100 Hz) and averaged (100 sums for each trial, excluding the events with artifacts). The analysis time was 500 ms.

Every trial was repeated at least twice and the resulting waveforms were superimposed to check for the repeatability of the results.

The transient response is characterized by a certain number of waves with three peaks, of negative, positive, negative polarity, respectively. In normal subjects these peaks have the following latencies: 75, 100 and 145 ms. If the control VEP of both the left and the right eye was normal, a second control VEP was recorded, reducing the averages to 40 events per trial (with no more than 2 sweeps discarded because of artifacts). This VEP record was defined as "basal" and kept on display on the computer screen.

Arterial pressure and IOP were then measured, the latter with the Perkins applanation tonometer.

Photostress was then induced in the left eye for 30 s by means of a circular diffusing surface (the bulb of a 200 W lamp) fixated by the subject form a distance of 20 cm and produced a central scotoma of 6 deg dia. Prior to the experimentation each subject was adapted to the ambient room lighting for 10 min, and the pupil diameter was about 3 mm. During the photostress procedure each subject looked directly into the center of the light, and the pupil diameter was about 1.5 mm. After dazzling, the subjects fixated a central point of the monitor and VEPs were then recorded (averaging over 40 stimulus

events) at 20, 40, 60, 80 s from the end of photostress.

A control and a "basal" VEP were then obtained for the right eye as described for the left eye.

After local anesthesia (novesine 0.4%) a Baillart ophthalmodynamometer was applied for 90 s to the right eye and the IOP was increased up to a value equal to half the systolic arterial pressure (conversion table). The systolic arterial pressure of the subjects varied between 120 and 140 mmHg and the IOP was increased according to values ranging between 60 and 70 mmHg.

The IOP was increased for a total period of 90 s. During the last 30 s a photostress was induced. Immediately after the end of photostress and increased IOP, fixation was shifted to the pattern stimulus and recording of VEPs started. Records were taken for successive periods of 20 s each (40 averaging every 20 s of recording and the corresponding record displayed on the screen) until the VEP obtained was superimposable to the control "basal" record. The corresponding time was considered the "recovery time after photostress".

For both VEPs, the peak latency and the peak amplitude of each wave were measured directly on the displayed records by means of a pair of cursors.

At the end of VEP recording session, the IOP was again measured.

RESULTS

In order to study macular function recovery after dazzling we have considered the results from:

Table 1. Analysis of variance

Latency P100		
Group	F (1,28)* = 33.7;	$P < 0.0000$
Treatment	F (3,84) = 266.6;	$P < 0.0000$
Group by treatment	F (3,84) = 55.3;	$P < 0.0000$
Temporal difference N75/N145		
Group	F (1,28) = 1.6;	$P = 0.22$
Treatment	F (3,84) = 38.2;	$P < 0.0000$
Group by treatment	F (3,84) = 1.47;	$P = 0.22$
Amplitude N75–P100		
Group	F (1,28) = 2.17;	$P = 0.15$
Treatment	F (3,84) = 35.6;	$P < 0.0000$
Group by treatment	F (3,84) = 3.58;	$P = 0.017$
Amplitude P100–N145		
Group	F (1.28) = 1.4;	$P = 0.24$
Treatment	F (3,84) = 47.4;	$P < 0.0000$
Group by treatment	F (3,84) = 3.32;	$P = 0.023$

*Degrees of freedom (d.f.).

—VEP after photostress in normal condition (LE);

—VEP after photostress in condition of artificially increased IOP (RE).

In the analysis of VEP records we have evaluated the P100 latency, the temporal difference N75/N145, the N75–P100 amplitude, and the P100–N145 amplitude.

The significancy of the differences between the two eyes were evaluated with two-way analysis of variance for repeated measures (Table 1).

VEP after photostress

Examples of records of one subject (LE) are shown in Fig. 1. The mean data are presented in Table 2 and Fig. 2 (solid bars).

The parameters of the basal VEP were within normal limits (P100 latency mean value 93.19 ± 3.2 ms; upper limit, mean + 3 SD = 102.08 ms).

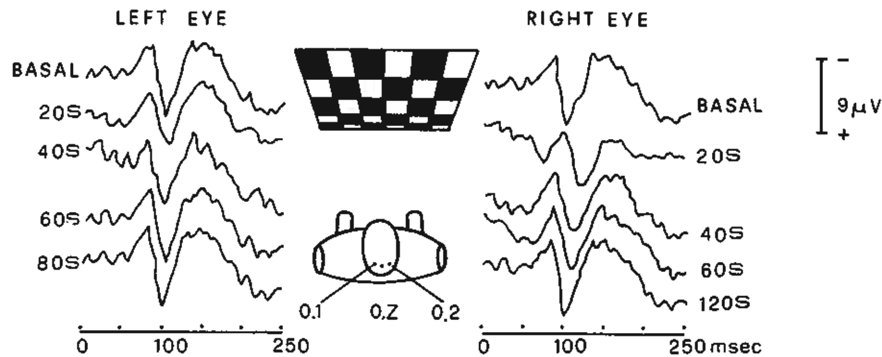

Fig. 1. VEP layout of subject SL in normal condition (basal) and 20, 40, 60, 80 and 120 s after photostress, LE is control eye, RE is eye with increased IOP. In comparison with the LE records, the RE VEPs recorded at 20, 40 and 60 s after photostress show a longer P100 latency and a reduced amplitude. The VEP was superimposable to the basal waveform at 80 s in the LE and at 120 s in the RE.

Table 2. Mean and standard deviation (±) of parameters of VEPs records in basal condition and 20, 40, and 60 s after photostress

	Latency P100 (ms)	Difference N75/N145 (ms)	Amplitude N75–P100 (μV)	Amplitude P100–N145 (μV)
Basal				
LE	93.19 ± 3.20	46.53 ± 3.10	9.02 ± 2.24	9.63 ± 2.77
RE	93.74 ± 3.43	48.74 ± 4.77	8.73 ± 2.12	9.59 ± 2.72
20s				
LE	105.10 ± 3.14	51.62 ± 5.43	7.45 ± 2.15	7.21 ± 1.88
RE	125.7 ± 8.14	54.96 ± 6.21	5.63 ± 1.45	5.61 ± 1.33
40s				
LE	101.66 ± 3.62	50.62 ± 4.63	7.64 ± 1.66	7.93 ± 2.32
RE	111.37 ± 7.47	51.75 ± 5.43	6.41 ± 1.69	6.55 ± 1.82
60s				
LE	97.72 ± 3.82	47.98 ± 3.05	7.98 ± 2.59	8.46 ± 2.65
RE	103.85 ± 5.36	49.44 ± 5.35	7.16 ± 1.60	7.88 ± 2.34

Left eye, control eye; right eye, eye with experimental increased IOP.

At 20 s after photostress, we observed an increase of the P100 latency (mean 105.10 ± 3.14 ms) and of the temporal difference N75/N145 (basal value, 46.53 ± 3.1 ms; value 20 s after photostress, 51.62 ± 5.43 ms).

At 40 and 60 s after photostress the P100 latency and the time difference N75/N145 decreased with respect to the values observed at 20 s, but were still higher than the basal ones. After 80 s from photostress the record was superimposable on the basal one, indicating perfect recovery. Neither the N75–P100 amplitude nor the P100–N145 amplitude, change after photostress.

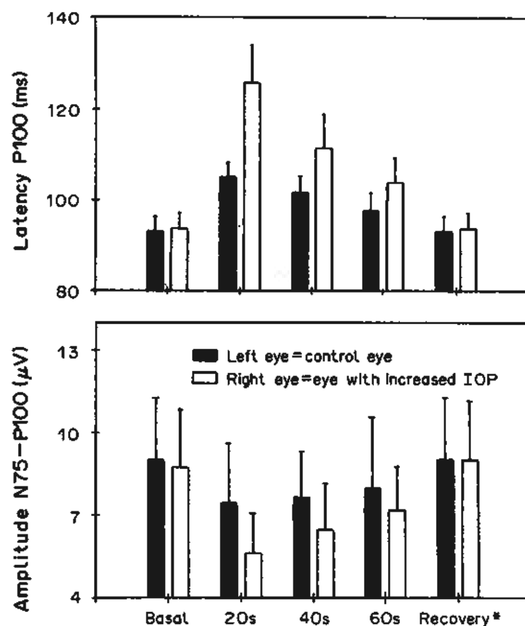

Fig. 2. Histograms of mean values of parameters of VEP in basal condition and 20, 40 and 60 s, after photostress (solid bars for control eye and open bars for the eye with increased IOP). The vertical lines represent standard deviation. *Recovery time after photostress was 80 s for the left eye and 114 s for the right eye.

VEP after photostress with artificially increased IOP

Examples of records of one subject (RE) are shown in Fig. 1. The mean data are presented in Table 1 and Fig. 2 (open bars).

Basal VEP values were within normal limits in all the subjects examined.

At 20 s from photostress and with artificially increased IOP we observed a change of all VEP parameters: the P100 latency increased from 93.74 ± 3.43 to 125.75 ± 8.14 ms; the difference N75/N145 increased from 48.74 ± 4.77 to 54.96 ± 6.21 ms; VEP amplitude decreased from 9.63 ± 2.7 to 5.61 ± 1.3 μV.

At 40 and 60 s from photostress the P100 latency and the N75/N145 difference progressively diminished still remaining, however, higher than under normal IOP. The VEP amplitude remained lower than normal. The VEP recovery time was in this group 114.2 ± 5.11 s, that is much longer than in the control group.

The analysis of variance revealed significant differences between the two eyes in both latency and amplitude VEPs. In particular in the eye with increased IOP, the P100 latency was delayed (main effect, $P < 0.0000$) and its recovery was slower in comparison with the eye with photostress only. In addition the VEP amplitude (either N75–P100 and P100–N145) showed a slower recovery (interaction effect, $P = 0.017$ and $P = 0.023$ respectively).

At 120 s from the application of the ophthalmodynamometer, the ocular tension had decreased compared to the mean basal values

(13.06 ± 0.8 mmHg), reaching a value of 5.73 ± 1.3 mmHg.

DISCUSSION AND CONCLUSIONS

The aim of our research was to evaluate the role played by the increase of IOP on the functional recovery capacity of the macula after dazzling.

In the control group, subjected to photostress, the VEP recorded 20 s after dazzling presented a latency increase and an amplitude decrease. After 80 s a normal condition was reached, and therefore the functional recovery, was complete.

We have not recorded the FERG after photostress in the control group. It has to be noted, however that the changes induced by photostress on VEPs are generally attributed to the diminished capacity of photoreceptors to produce a sufficient electrotonic potential after dazzling (Lovasik, 1983; Franchi *et al.*, 1987).

In the eyes with artificially increased IOP the parameters of VEP after photostress undergo larger changes than the control group. This is attributed to the hypertension and could also be related to a further suffering of the photoreceptors or to suffering of the ganglion cell layer.

Previous reports (Karpe, 1945; Henkes, 1951; Iser and Goodman, 1956; Vanysek, 1956; Francois and De Roux, 1959; Busti, 1962; Ponte, 1962; Wanger and Persson, 1983; Marx *et al.*, 1986a,b) indicate that the FERG is not modified after ocular hypertension or in glaucoma: this leads us to believe that the sensorial layer of the retina is not functionally sensitive to the amount of intraocular pressure.

Evidence that ocular hypertension mainly affects the proximal retinal layers came from a recent work in humans which shows that experimentally induced ocular hypertension produced modifications of VEP and PERG but not of FERG (Kothe and Lovasik, 1989; Lovasik and Kothe, 1989).

All this is supported by an experimental model of retinal ischemia in the cat (Siliprandi *et al.*, 1988), here the IOP was increased and the average arterial pressure was reduced until a total block of the coroid–retina circulation was obtained. After 10 min from ischemia, a prompt recovery of FERG was found, while the VEP and PERG were found to be still depressed, even 2 hr from the ischemia. This fact reveals that the neurons of the proximal layers are more vulnerable to ischemic damage than those of the distal layers.

When hypertension is present, the longer VEP recovery after macular dazzling (on average 114 s) can be explained by stress caused by the ischemia of the macular ganglion cells and of the papillo-macular bundle axons. In fact the macular axons form neural fibers that travel inside the optic nerve and are "densely packed". They are very thin and have a very high metabolism. Moreover, as they are irrigated by a very delicate capillary system, they are very sensitive to ischemic damage (Lindberg and Walsh, 1964; Potts *et al.*, 1972).

The decrease in ocular tone at the end of examination does not seem to have influenced the VEP. This agrees with a previous study (Bucci *et al.*, 1988) in which it was found that a hypotension of 3–4 mmHg for the duration of 20 min did not give way to any VEP changes.

Our results indicate that the VEP recovery time after photostress depends on retinal photopigment ability of resynthesis and on trophism of the macula papillo-macular bundle system.

Therefore this kind of electrophysiological test offers a possible application for the diagnosis of macular pathology and for the clinical evaluation of the functional state of the papillomacular bundle, in pathologies like glaucoma in which this structure undergoes anatomical and pathological changes, which produce a functional deficit.

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